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Contrast-induced acute kidney injury in patients submitted to coronary angioplasty: prospective cohort

Lesão renal aguda induzida por contraste em pacientes submetidos à angioplastia coronariana: coorte prospectiva

Lesión renal aguda inducida por contraste en pacientes sometidos a angioplastia coronaria: una cohorte prospectiva

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ABSTRACT

Objective: To analyze the incidence, risk factors, and associations of clinical outcomes for contrast-induced acute kidney injury (CI-AKI) in patients with acute coronary syndrome (ACS) after coronary angioplasty. **Method:** Prospective cohort of 182 patients followed for three months after undergoing angioplasty, from July 2020 to June 2021. The analyzed variables were sociodemographic, clinical, and those related to the procedure. **Results:** The incidence of CI-AKI was 35.7% (n = 65) and was associated with old age, diabetes mellitus, and chronic kidney disease (p = 0.004, p < 0.001, and p = 0.009, respectively). Out of the 17 patients who died within 90 days, 76.5% had CI-AKI (n = 13), the odds ratio between death and CI-AKI was approximately 7.2 times (95% confidence interval (CI), [2.41;26.36]; p = 0.001). The decrease of one unit in the patient's baseline hemoglobin showed a 6.5% increase for CI-AKI (95% CI, [−0.089; −0.040]; p < 0.0001). **Conclusion:** CI-AKI is prevalent in patients with ACS after angioplasty and is related to diabetes mellitus and chronic kidney disease, showing high mortality rates.

DESCRIPTORS

Kidney Diseases; Acute Kidney Injury; Contrast Media; Percutaneous Coronary Intervention; Acute Coronary Syndrome.

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INTRODUCTION

The advance of Western medicine and the technological contributions of recent decades have created a therapeutic model for the treatment of chronic arterial diseases: the percutaneous coronary intervention (PCI). This model has become widely used as an alternative to drug therapy and surgical coronary revascularization, emerging as a practical, agile, and less-invasive alternative with lower rates of postoperative complications and mortality[1]. However, despite being an excellent therapeutic model, PCI—also known as coronary angioplasty—also offers a potential risk associated with exposure to iodinated contrast (IC) media[2].

Notably, iodine and other contrasting agents can improve the definition of tomographic and radiological images, conducting and assisting medical diagnoses and interventions[3]. The substance is widely used in daily clinical-hospital practice. However, endovascular contrasting agents are known as potential causes of acute kidney injury (AKI), also known as contrast-induced nephropathy or contrast-induced acute kidney injury (CI-AKI)[2,4,5].

Though the pathogenesis of CI-AKI is still partially undetermined, experimental studies suggest that contrast affects renal vascular tonicity, producing an endothelial vasodilator effect; vasoactive agents adenosine and endothelin then induce vasoconstriction, which causes endothelial dysfunction, inflammation, toxicity, and cellular apoptosis, resonating in tissue hypoxia, oxidative damage, and hemodynamic changes[4–6]. In the hospital scope, CI-AKI is the third leading cause of acute kidney injury[5–7].

Considering that contrast-enhanced tests are essential for the diagnosis and clinical management of acute coronary syndrome (ACS) and that the renal system is a vulnerable adjuvant when associated with clinical comorbidities, this study aimed to analyze the incidence, risk factors, and associations of clinical outcomes for CI-AKI in patients with ACS subjected to angioplasty.

METHOD

Type of Study

This is a prospective cohort study of quantitative approach developed by structured interview and data analysis in medical records.

Population, Location, and Inclusion Criteria

The study included 242 patients who underwent PCI for ACS and were admitted to the hemodynamics unit of a university hospital which provides services to the Brazilian Unified Health System (SUS) and to previously registered health insurance plans, being a reference for medium and high complexity care in the state of São Paulo. The patients referred to the hemodynamics service came from other hospitals or from hospitalization units or intensive care unit (ICUs) of the university hospital itself. Out of the 242 patients, 182 were eligible, including those aged over 18 years, diagnosed with ACS, who underwent PCI at the described site, remained hospitalized for over 24 hours, presented pre- and post-examination serum creatinine values, and signed the informed consent form.

Data Collection

Data was collected daily via Research Electronic Data Capture (REDCap), an electronic platform for data collection and management, by four research students who approached patients or family members for research participation from July 2020 to June 2021. After the patients signed the consent form, the students interviewed them to validate and to complement their clinical and demographic data. Data entered in the worksheet was double-checked and an audit was conducted to reconfirm data tabulation. The variables collected were sociodemographic, clinical, and laboratory, namely: gender; age; skin color; schooling level; professional occupation; body mass index (kg/m^2); previous chronic comorbidities; smoking (years/pack); Killip classification; left ventricular ejection fraction (LVEF); electrocardiographic changes and heart rhythm; medications in use; previous heart failure (HF); previous cerebrovascular accident (CVA); and history of acute myocardial infarction (AMI), PCI, or previous surgical myocardial revascularization. The following exams were serum creatinine (SCr), serum hemoglobin, serum potassium, troponin T, and reverse transcription polymerase chain reaction (RT-PCR) for COVID-19 detection. Data regarding PCI were also used, including the type of contrasting agent used, contrast-infused volume, evaluated artery, number of stents used, dose of heparin administered, and total time of examination.

Three collections were performed for laboratory tests of SCr, serum hemoglobin, serum potassium, and troponin T. The first collection was performed one day before PCI, the second on the second day after PCI, and the third collection on the third day after PCI.

All patients were assessed at the time of admission to the hemodynamics sector and then followed for 90 days after PCI. Their subsequent information were obtained from outpatient consultations, hospital visits, hospital admissions, and/or by telephone. CI-AKI was considered as the main outcome and dependent variable. The secondary outcomes analyzed were death, dialysis, and attendance to the emergency room for signs, symptoms, or clinical complications.

Definitions

CI-AKI was considered as a ≥0.5 mg/dl increase in SCr or a relative increase of ≥25% from the baseline 48 h to 72 h after intervention according to Kidney Disease: Improving Global Outcomes – KDIGO[7]. Chronic Kidney Disease (CKD) was defined as the decrease in glomerular filtration rate to below 60 ml/min/1.73 m^2 and/or the presence of abnormalities in the renal structure which last for over three months[7].

For ST-elevation myocardial infarction (STEMI) or non-ST-elevation myocardial infarction (NSTEMI), the clinical context of ischemia was considered for increased levels of necrosis biomarkers and the existence or not of electrocardiographic alterations. Clinically, new significant changes in the ST segment, T wave, or new left bundle branch block are associated with typical precordial chest pain, evidence of

myocardial loss, alteration of new ventricular contractility, visualization of thrombi, or identification, by angiography, of severe intracoronary lesions. Specifically for STEMI, in short, ST elevation ≥1 mm in two or more contiguous leads corresponding to the lesion area was considered. For NSTEMI, the presence of AMI was considered with no elevation of the ST segment and possible alterations of ST, such as segment depression, or of the T wave. For unstable angina, the clinical context with negative necrosis biomarkers was considered, with possible electrocardiographic alterations[8].

PCI Protocol

Percutaneous Coronary Intervention was performed by proximal radial, distal, or femoral access using introducers from 5F to 6F, therapeutic catheters, and conventional and pharmacological *stents*, with pre- or post-intracoronary balloon dilatation during stent implantation. Stent implantation was followed by ventriculography. For PCI, unfractionated heparin (100 IU/kg) was used, with protamine sulfate reversal at the end of the procedure and non-ionic contrast of low-osmolar iobitridol (henetix®) 300 mg/ml. To determine the angiographic success of the procedure, the Thrombolysis in Myocardial Infarction (TIMI) flow classification[9] was used, considering four flow levels: the lowest, Grade 0, if the anterograde flow goes up to the occlusion point; and the highest, Grade 3, complete coronary perfusion with anterograde flow in the distal bed, occurring promptly as in the proximal coronary bed. In this cohort, only patients with TIMI score 3 were selected.

Data Treatment and Analysis

The data obtained were saved on the REDCap electronic platform and transferred to a spreadsheet in the Excel-2016 program from Microsoft® Windows. They were then analyzed by software R® version 4.0.4 with the packages MANOVA.RM, rstatix, hnp, nnet, and openxlsx. Descriptive analysis was performed for all study variables in order to characterize the sample. The qualitative variables were described by absolute (n) and relative (%) frequencies. For the quantitative variables, mean and standard deviation were calculated. Differences in proportions were assessed by chi-square test and Fisher's exact test. Student's *t*-test and Mann-Whitney U test were used to compare continuous variables in normal and abnormal distributions. For the logistic model, the variable of interest was CI-AKI, analyzed categorically (present or not present). The Hosmer-Lemeshow test was used to verify the suitability/validity of the model, appropriating the null hypothesis as an appropriate model and the alternative hypothesis as an inadequate model (significance level = 5%; p-value = 0.091). The ROC curve, used as a discriminatory model of individuals, had value 0.68, indicating discrimination close to the reasonable (70%). The odds ratio (OR) and their respective confidence intervals (95% CI) were presented to quantify the effects.

Ethical Aspects

The research was analyzed and approved in 2020 by the Research Ethics Committee of the Universidade Federal de São Paulo under opinion No. 3.763.447 according to Brazilian resolution 466/12.

RESULTS

In total, 182 out of 242 ACS patients included in the study were eligible for participation (Figure 1).

Out of the 182 patients, 65 (35.7%) had CI-AKI incidence. Among participants, the mean age was 62.03 ± 10.50 years, 71% (n = 130) were men, 66% (n = 121) self-declared as White, 27% (n = 50) did not finish elementary school, and 36% (n = 65) were retired. The main clinical comorbidities of our sample's patients were: systemic arterial hypertension (SAH) – 64.8% (n = 118), dyslipidemia – 45.1% (n = 82), diabetes mellitus (type 2) – 28.6% (n = 52), and thyroid disease and CKD – 8.2% (n = 15) each. Regarding risk factors, we found that 68.7% participants (n = 125) were smokers, 43.4% (n = 79) were obese, 20.9% (n = 38) had AMI history, and 48.9% (n = 89) had LVEF

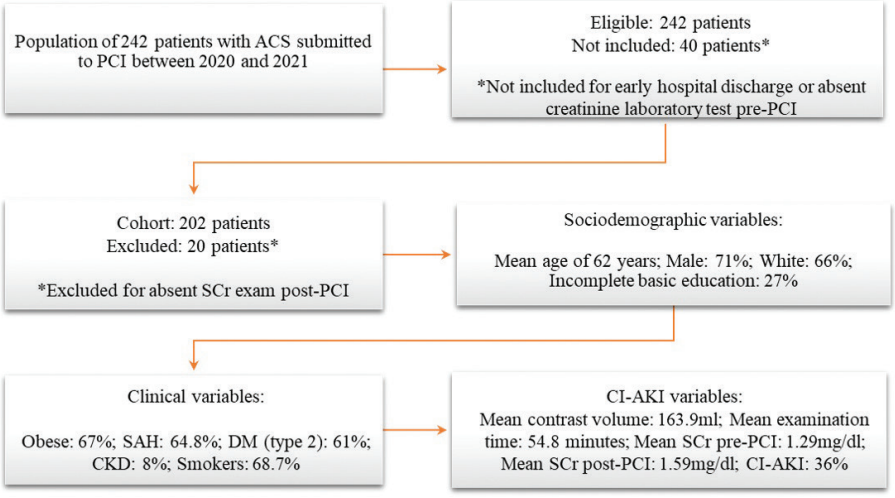

Figure 1 – Description of the flow of inclusion of people in the study and general description of the sample – São Paulo, Brazil, 2021.

PCI – Percutaneous coronary intervention; ACS – Acute Coronary Syndrome; SCr – Serum Creatinine; SAH – Systemic Arterial Hypertension; DM – Diabetes Mellitus; CKD – Chronic Kidney Disease; CI-AKI – Contrast-Induced Acute Kidney Injury.

Table 1 – Sociodemographic and clinical variables of patients – São Paulo, SP, Brazil, 2021.

CHARACTERISTIC	No CI-AKI group (n = 117)	%	Mean (SD)	CI-AKI group (n = 65)	%	Mean (SD)	ALL PATIENTS (n = 182)	Mean (SD)	p-value
Men	89	68.5		41	31.5		130		0.063**
Age			**60.2 ± 10.5**			**64.9 ± 9.9**		**62.0 ± 10.5**	**0.004*** **
White	76	62.8		45	37.2		121		0.763*
Incomplete elementary education	26	52.0		24	48.0		50		0.411*
Retired	36	55.4		29	44.6		65		0.725*
SAH	72	61.0		46	39.0		118		0.498*
Type 2 DM	**25**	**48.1**		**27**	**51.9**		**52**		**<0.001****
Dyslipidemia	55	67.1		27	32.9		82		0.477**
CKD	**5**	**33.3**		**10**	**66.7**		**15**		**0.009****
Stage 5 CKD	**1**	**12.5**		**7**	**87.5**		**8**		**0.003***
Thyroid disease	10	66.7		5	33.3		15		0.841**
Previous AMI	25	65.8		13	34.2		38		0.828**
Previous surgical revascularization	4	44.4		5	55.6		9		0.285*
Previous PCI	17	63.0		10	37.0		27		0.876**
Previous CVA	4	100.0		0	0.0		4		0.298**
Smoking History	85	68.0		40	32.0		125		0.121**
Smoking in Years/Pack									
≤ 40	39	68.4		18	31.6		57		
41–80	22	59.5		15	40.5		37		0.705*
81–120	5	71.4		2	28.6		7		
> 121	1	33.3		2	66.7		3		
Body mass index from 25 to 29.9 kg/m²	53	67.1		26	32.9		79		0.597*
LVEF ≤ 55 (%)	53	59.6		36	40.4		89		0.279**
Previous HF	12	60.0		8	40.0		20		0.672**
STEMI diagnosis	63	67.0		31	33.0		94		0.619**
Killip II	8	44.4		10	55.6		18		0.188*
Previous beta-blocker use	31	57.4		23	42.6		54		0.208**
Previous use of loop diuretics	5	41.7		7	58.3		12		0.119*
Previous use of ACEI	28	58.3		20	41.7		48		0.316**
Previous use of ARB	31	64.6		17	35.4		48		0.96**
Previous use of thiazide diuretics	21	77.8		6	22.2		27		0.113**
Previous use of CCB	14	60.9		9	39.1		23		0.714**
Previous use of potassium-sparing diuretics	**4**	**36.4**		**7**	**63.6**		**11**		**0.050***
Previous use of statins	44	67.7		21	32.3		65		0.475**

CI-AKI – Contrast-Induced Acute Kidney Injury; SAH – Systemic Arterial Hypertension; DM – Diabetes Mellitus; CKD – Chronic Kidney Disease; AMI – Acute Myocardial Infarction; PCI – Percutaneous Coronary Intervention; CVA – Cerebrovascular Accident; LVEF – Left Ventricular Ejection Fraction; HF – Heart Failure; STEMI – ST-elevation myocardial infarction; ACEI – Angiotensin-Converting Enzyme Inhibitor; ARB – Angiotensin Receptor Blocker; CCB – Calcium Channel Blocker; *Fisher's exact test. **Chi-square test. ***Student's t-test.

lower than 55%. The main diagnosis of patients was STEMI [51.6% (n = 94)] and 9.8% (n = 18) were classified as KILLIP II.

Table 1 shows that, among groups without CI-AKI and with CI-AKI, variables with statistical significance were age (p = 0.004), Type 2 diabetes mellitus (p < 0.001), CKD (p = 0.009), stage 5 CKD (p = 0.003), and previous use of potassium-sparing diuretics (p = 0.05).

Regarding the variables related to PCI and CI-AKI (Table 2), we found that the anterior descending coronary artery approach was used in 49.5% (n = 90) of the cases. Although patients with CI-AKI had higher baseline SCr numerically, they had no statistically significant differences of mean SCr with patients without CI-AKI (1.0 ± 0.4 vs 1.7 ± 1.8, respectively; p = 0.906). On the other hand, serum creatinine increased significantly between the two groups after clinical examination (1.0 ± 0.4 vs 2.5 ± 2.2, respectively p < 0.001). The mean volume of infused contrast was 163.9 ± 61.197 and the mean duration of the procedure was 54.8 ± 26.834. The group of patients without CI-AKI received a higher dose of heparin during PCI than the group with CI-AKI (8158.73 ± 8821.2 vs 7845.69 ± 1553.5; p = 0.023). Serum hemoglobin significantly decreased in the CI-AKI group (p = 0.002).

Table 2 – Variables related to PCI and clinical examinations – São Paulo, SP, Brazil, 2021.

VARIABLES	No CI-AKI group (n = 117)	%	Mean (SD)	CI-AKI group (n = 65)	%	Mean (SD)	ALL PATIENTS (n = 182)	Mean (SD)	p-value
Positive RT-PCR for COVID-19 during PCI	3	42.9		4	57.1		7		0.250*
PCI time			52.4 ± 23.3			59.3 ± 32.1		54.6 ± 26.8	0.203#
No. of stents used = 1	74	66.1		38	33.9		112		0.574*
Addressed artery LAD	54	65.9		28	34.1		84		0.688*
Mean baseline SCr			1.0 ± 0.4			1.7 ± 1.8		1.3 ± 1.2	0.906#
Heparin dose during PCI (ml)			**8158.7 ± 8821.2**			**7845.7 ± 1553.5**		**7956.0 ± 5355.8**	**0.023 #**
Contrast volume 101–200ml	83	67.5		40	32.5		123		0.205*
Post-PCI SCr			**1.0 ± 0.4**			**2.5 ± 2.2**		**1.5 ± 1.5**	**<0.001#**
Serum potassium 48h after PCI			4.268 ± 0.659			4.405 ± 0.737		4.316 ± 0.689	0.183#
Peak Troponin			3442.0 ± 5544.8			6496.6 ± 13578.69		4543 ± 9356.018	0.277#
Serum hemoglobin			**14.3 ± 2.3**			**13.1 ± 2.5**		13.9 ± 2.5	**0.002 #**

CI-AKI – Contrast-Induced Acute Kidney Injury; RT-PCR – Reverse Transcription Polymerase Chain Reaction; PCI – Percutaneous Coronary Intervention; LAD – Left Anterior Descending Artery; SCr – Serum Creatinine. * Fisher's exact test. # Mann-Whitney U test.

Table 3 – Association between secondary outcomes after 30 and 90 days and occurrence of CI-AKI – São Paulo, SP, Brazil, 2021.

Secondary outcome	WITHOUT CI-AKI	%	WITH CI-AKI	%	ALL PATIENTS	p-value
Death						
No	113	68.5	52	31.5	165	**0.003****
Yes	4	23.5	13	76.5	**17**	
In 30 days						
Emergency room care	10	71.4	4	28.6	14	
Dialysis and death	0	0.0	1	100.0	1	0.123*
Dialysis	1	33.3	2	66.7	3	
Death	2	25.0	6	75.0	8	
90 days						
Emergency room care	7	77.8	2	22.2	9	
CKD	1	50.0	1	50.0	2	
CVA	1	50.0	1	50.0	2	**0.049***
AMI	1	100.0	–	–	1	
Death	1	50.0	1	50.0	2	

CI-AKI – Contrast-Induced Acute Kidney Injury; CKD – Chronic Kidney Disease; CVA – Cerebrovascular Accident; AMI – Acute Myocardial Infarction. * Fisher's exact test. ** Chi-square test.

Table 4 – Logistic model and odds ratio for CI-AKI occurrence – São Paulo, SP, Brazil, 2021.

Logistic model	Estimate	Mean (SD)	95%CI of parameters	Pr(>|z|)
Death	1.969	0.595	[0.881; 3.272]	**0.001**
Pre-PCI serum hemoglobin	–0.063	0.012	[–0.089; –0.040]	**<0.001**
Odds ratio (OR)	**OR**		**95%CI of odds ratio**	**p-value**
Death	7.164		[2.414; 26.365]	**0.001**
Pre-PCI serum hemoglobin	0.939		[0.915; 0.961]	**<0.001**

PCI – Percutaneous Coronary Intervention.

Our sample's main outcomes were the need to attend the emergency room, rehospitalizations, and death. Around 9.34% of the sample's patients died (n = 17), of which 7.14% (n = 13) had CI-AKI; therefore, 76.5% of all deaths were classified as CI-AKI (Table 3). For the odds ratio model, those who die are about 7.2 times more likely to have been classified with CI-AKI than patients who do not die (odds ratio, 7.16; 95% CI, [2.41;26.36]; p = 0.001). Moreover, each decreased unit in pre-PCI hemoglobin increases by 6.3% the chance of having CI-AKI as the main outcome. (Logistic model estimate, -0.063; SD, 0.012; 95% CI, [-0.089; -0.040]; Pr(>|z|) <0.0001) (Table 4).

DISCUSSION

This study showed a factual incidence of CI-AKI in about one third of patients with ACS who underwent coronary angioplasty in a university hospital, with data different from the Brazilian study described by Santos et al. for the same group of patients, which showed a 23.8% incidence of CI-AKI[10]. CI-AKI incidence varies according to risk profile and procedure modality and can be estimated from 1 to 90%, in which a high incidence is related to CKD and DM[11,12]. A recent meta-analysis showed that in patients with ACS, the incidence ranges from 4.7% to 35.7%[13].

CI-AKI incidence also varies with type and volume of contrast infused, conditions for performing the procedure, and even patient's clinical characteristics. Some authors have already elaborated an important risk stratification score using variables that expanded CI-AKI development, including: age >75 years, SAH, dyslipidemia, diabetes, severe heart failure of functional class III/IV, ACS, CKD, anemia, multivessel disease, hypotension, use of intra-aortic balloon, type of iodinated contrast, and infused contrast volume[11]. Our study showed that old age was characterized as an adjunct to statistically significant variables. Recent studies relate this to the fact that older adults are more likely to have the score risk factors[3,11,12]. CKD, DM, and anemia were also significant, corroborating current concrete scientific evidence[5,11,12,14–16]. By logistic regression model and odds ratio, we found that each decreased unit of pre-PCI serum hemoglobin increased by 6.5% the chance of having CI-AKI. This data corroborates the findings of a Chinese study, which found that a patient's baseline hemoglobin was an independent predictor for CI-AKI[15], and an English study, which showed that the lower baseline hematocrit was also a predictor of CI-AKI[17]. Each 3% decrease in baseline hematocrit significantly affects the incidence of CI-AKI[17].

The renal medulla region, which contains renal tubules, is physiologically characterized by its activity under hypoxic regime due to the intrarenal physiological gradient of oxygen. Thus, a higher oxygen consumption by renal tubular transport of chloride and sodium, that is, a high metabolic activity, would increase susceptibility to kidney ischemia[15]. Medullary oxygenation is therefore physiologically low and more sensitive and vulnerable to hypoxia. Accordingly, studies show that intravascular contrast can increase hemoglobin affinity for oxygen, impairing tissue oxygenation[14]. The vasoconstrictive effects of iodinated contrast exposure associated with low hemoglobin concentration could thus decrease the supply of medullary oxygen, promoting renal medullary hypoxia. Anemia is therefore a significant risk factor for CI-AKI.

Moreover, the mean dose of fractionated heparin in milliliters used for each of the two groups was different. The group of patients without CI-AKI received a higher dose of heparin. A recent study found that antithrombin III (ATIII), a physiological influencer of the endogenous anticoagulation process—which can be intensified by substances such as heparin. It is a significant protective factor for CI-AKI[18], showing that patients with low ATIII activity have a higher incidence of renal injury after cardiac catheterization or PCI[18,19]. This may be related to the anti-inflammatory factor of this endogenous molecule. Furthermore, the use of ATIII enhancers still has no significant association with CI-AKI. Studies have focused on the predictive value of baseline ATIII serum levels and the substance supplementation as prophylaxis[18–20].

Regarding contrast volume, several authors have associated the use of lower contrast volumes with a reduced risk for CI-AKI[5,21–23]. The high concentration of serum contrast can stimulate its deposit in the renal tubules and increase tubular pressures, decreasing the glomerular filtration rate and renal blood flow and increasing oxygen consumption and metabolic activity by osmotic diuresis[24]. Our study showed that patients with CI-AKI mostly received a mean 100–200 ml volume of infused contrast. Low-osmolar non-ionic iobitridol contrast was used to reduce the agent "osmotoxicity" in the intravascular environment and the nephrotoxic potential of the most potent contrasting agents.

Regarding the use of different contrasting agents, used to reduce kidney damage, robust and well-disseminated evidence shows that high-osmolar contrast media have higher nephrotoxic potentials than low- and iso-osmolar contrasts[25–26]. Some experimental and clinical studies found that the use of iso-osmolar contrast is significantly more favorable than low-osmolar contrast; however, the former's clinical superiority over the latter is still undefined[25–27]. Considering these controversial results, cautiousness is recommended regarding the infused volume of contrast, whether in low or iso-osmolarity media[24].

This cohort found that 7.14% of patients with CI-AKI died, and those who die are 7.2 times more likely to be from the CI-AKI group than from the group without CI-AKI. Many studies associate this comorbidity with a significant

increase in inpatient dialysis and deaths, with data showing 36% hospital mortality and 19% survival in two years[14,28]. Researchers are currently discussing whether the hemodynamic state of the patient is the core point of the worst outcomes and mortality from CI-AKI. However, studies indicate CI-AKI as an independent predictor for long-term mortality, even after sample correction for hemodynamically stable patients with preserved LVEF[29]. CI-AKI thus interferes with inpatient mortality and is directly related to the elevation of laboratory parameters of serum creatinine[15].

In this study, the proposed methodological design has relevant theoretical limitations—especially from the conclusive point of view, in which the validity of correlations in the sample can be identified without identifying the causes. Moreover, the reduced sample size can affect statistical inference, overshadowing the analyses of significant and predictor associations. A longer follow-up period could have found late clinical outcomes, clarifying the relationships between CI-AKI outcome and morbidity and mortality. Developing new research aimed at prophylactic practices for CI-AKI is therefore essential considering the higher incidence of the injury in patients with ACS.

CONCLUSION

CI-AKI affected about one third of the sample and factors such as DM, CKD, and serum hemoglobin levels were significant for this outcome. The decrease in serum hemoglobin pre-PCI is proportional to the increased chance of having nephropathy. Patients who died at the end of follow-up were associated with CI-AKI.

RESUMO

Objetivo: Analisar a incidência, os fatores de risco e as associações dos desfechos clínicos para Lesão Renal Aguda Induzida Por Contraste (LRA-IC) em pacientes com Síndrome Coronariana Aguda (SCA) após angioplastia coronariana. **Método:** Coorte prospectivo de 182 pacientes seguidos por três meses após angioplastia, entre julho de 2020 e junho de 2021. As variáveis foram sociodemográficas, clínicas e relacionadas ao procedimento. **Resultados:** A incidência de LRA-IC foi de 35,7% (n = 65) e esteve associada à idade avançada, diabetes mellitus e doença renal crônica (respectivamente p = 0,004, p < 0,001 e p = 0,009). Dos 17 pacientes que faleceram em até 90 dias, 76,5% tiveram LRA-IC (n = 13), a razão de chances entre óbito e LRA-IC foi de aproximadamente 7,2 vezes (intervalo de confiança (IC) 95%, [2,41;26,36]; p = 0.001). A diminuição de uma unidade na hemoglobina basal do paciente demonstrou um aumento de 6,5% para LRA-IC (IC 95%, [–0,089; –0,040]; p < 0,0001). **Conclusão:** Em pacientes com SCA após angioplastia, a LRA-IC tem alta incidência e está relacionada com diabetes mellitus e doença renal crônica, apresentando altos índices de mortalidade.

DESCRITORES

Nefropatias; Injúria Renal Aguda; Meios de Contraste; Intervenção Coronária Percutânea; Síndrome Coronariana Aguda.

RESUMEN

Objetivo: Analizar la incidencia, los factores de riesgo y las asociaciones de resultados clínicos para Lesión Renal Aguda Inducida por Contraste (LRA-IC) en pacientes con Síndrome Coronario Agudo (SCA) después de angioplastia coronaria. **Método:** Cohorte prospectiva de 182 pacientes monitorizados durante tres meses tras realizada la angioplastia, en el periodo entre julio de 2020 y junio de 2021. Se evaluaron las variables sociodemográficas, clínicas y relacionadas con el procedimiento. **Resultados:** La incidencia de LRA-IC fue del 35,7% (n = 65) y se asoció con la edad avanzada, la diabetes mellitus y la enfermedad renal crónica (p = 0,004, p < 0,001 y p = 0,009, respectivamente). De los 17 pacientes que fallecieron dentro de los 90 días, el 76,5% tenía LRA-IC (n = 13), la razón de probabilidad entre muerte y LRA-IC fue aproximadamente 7,2 veces (intervalo de confianza, IC del 95%, [2,41;26,36]; p = 0,001). La disminución de una unidad en la hemoglobina basal del paciente demostró un aumento del 6,5% para LRA-IC (IC del 95%, [–0,089; –0,040]; p < 0,0001). **Conclusión:** En los pacientes con SCA después de angioplastia, la LRA-IC tiene una alta incidencia y está relacionada con la diabetes mellitus y la enfermedad renal crónica, presentando altas tasas de mortalidad.

DESCRIPTORES

Enfermedades Renales; Lesión Renal Aguda; Medios de Contraste; Intervención Coronaria Percutánea; Síndrome Coronario Agudo.

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